supposed, an alkaloid, and for it the name *Strophantine* given to it some years ago by Dr. Fraser, is retained. Besides, they succeeded in isolating a substance presenting the characters of an alkaloid, but which did not seem to possess any marked physiological properties; for this they propose the name *Ineine.* The former is very poisonous, a single crystal placed under the skin of a frog's foot causing the cessation of the heart's action in a few moments. Even after this has taken place the animal still possesses the power of motion, and it is only after respiration has become impossible, owing to the interruption of circulation in the nervous centres, that death ensues from paralysis of the heart. These observations, though yet incomplete, accord pretty well with facts recorded by different authors, and seem to prove that *strophantine* is really the poisonous agent in *Strophanthus hispidus.* The most elaborate experiments on the poison found at the extremity of the arrows, (used by the natives, both in war and hunting) are those conducted by MM. Carville and Polaillon in the laboratory of M. Vulpian. They were made on various classes of animals, and show that the deadly action is much more rapid in mammals and birds than in mollusks, crustaceans, and fishes. On frogs under the influence of curare the poison acts much more slowly, though the respective actions of the two substances do not neutralize each other. *Nature*, Oct. 11, 1877.

THE POISONOUS PROPERTIES OF THE BULBOUS AGARIC.—At the séance of the Acad. de Médicine, Paris, Oct. 16, 1877, (rep. in *Gaz. des Hopitaux* No. 121) M. Gubler read for M. Oré some extracts from a paper on poisoning by the bulbous agaric, (*Amanita bulbosa*) the same being in support of the following propositions:

1. That the symptoms observed during life, in animals who were under the influence of this fungus, presented, in their convulsive phenomena, very great analogies with those produced by strychnia.

2. That the lesions observed at the autopsy, consisting in more or less pronounced congestions, with ecchymoses, ulcerations, etc., and occupying the whole extent of the gastro-intestinal mucous membrane, likewise offered a great resemblance to the lesions we observe in animals who have succumbed to the influence of that alkaloid.

3. Studying the manner in which the poisonous principle of the agaric and that of strychnia act in solution towards powdered carbon, M. Oré has shown that if we throw the two solutions on a filter after stirring them up with this powder the latter has the property of retaining both.

4. He finally seeks to prove that if we inject into the veins of one dog a solution of strychnia, and in another the acidulated water in which bulbous agarics have been macerated, death of both animals occurs with the same rapidity, and is preceded by absolutely similar symptoms.

THE ANTAGONISM OF ATROPINE AND MUSCARINE.—The following is a note communicated to the Acad. des Sciences, Paris, at the session of Oct. 8, 1877, by Mr. J. L. Prevost, of Geneva, as reported in the *Bull. Gen. de*

Thérapeutique, Oct. 30. "The experiments of which I give here the summary from part of a study on physiological antagonism, which I reported to the biological section of the International Congress for the Medical Sciences (Geneva, 5th session, Sept. 9–15) on the 14th of September last.

"It has been known, since the experiments of MM. Schmiedeburg and Kopp, that atropine is the antagonist and antidote to muscarine. In fact, atropine is not only capable of causing all the symptoms produced by muscarine to disappear, but this agent may be considered as its antidote, and it delays the death produced by a toxic dose of muscarine. My experiments have only confirmed, in this respect, the observations of MM. Schmiedeburg and Kopp, and others.

"The mutual antagonism of the two poisons has been denied almost up to the present, by various experimenters, who have all maintained that muscarine is without effect on animals who have received even the minimum dose of atropine.

"My experiments do not support this opinion, but show that large doses of muscarine are sufficient to produce toxic effects in animals previously atropinized. A number of the experiments consisted in injecting locally into the arteries of the sub-maxillary gland (procedure of M. Haidenhain) a strong dose of muscarine, in cats previously chloralized, and into the veins of which a dose of from one to five milligrammes of atropine had been injected.

"As soon as a dose of from 10 to 20 centigrammes of muscarine was reached I have seen a decided salivation produced just as if the animal had taken no atropine.

"In injecting muscarine into the peripheral end of a branch of the mesenteric artery, I have seen tetaniform vermicular contractions produced in the corresponding intestinal loop, in spite of the previous administration of atropine to the animals experimented upon (cats, rabbits, fowls, pigeons). This result was less constant and striking than was the salivation.

"In many of the experiments, the injection into the venous circulation of cats, previously atropinized, of large doses of muscarine produced the salivary, ocular, intestinal, and respiratory symptoms of the latter.

"In a cat, operated on July 31, 1877, I have seen the effect of muscarine shown twice in succession in spite of the previous injection of atropine; the second time the dose being as much as five milligrammes. Very large doses of muscarine are requisite to produce this result.

"Two milligrammes of sulphate of atropine, previously injected were neutralized by 76 centigrammes of muscarine.

"Five milligrammes of sulphate of atropine, injected into the same vein, suspended the action of the muscarine, which reappeared after an injection of 2.20 gr. of muscarine. The cat thus experimented upon, received altogether and in succession seven milligrammes of sulphate of atropine and three grammes of muscarine.

"This experiment was successfully repeated on other animals.

"These experiments permit me to conclude that the antagonism of atropine and muscarine is a mutual one, and that sufficiently large doses of

muscarine will produce the effects of the poison in animals previously atropinized, a fact that has hitherto been denied by experimenters.

"My experiments were made only in view of the antagonism of the two poisons, and do not permit me to affirm that muscarine in large doses is the antidote to atropine, as atropine is the antidote to muscarine even in feeble doses."

BROMIDE OF POTASSIUM.—G. Klosz. *Archiv. f. exper. Path.*, etc., VI. p. 1. (Abstr. in *Centralblatt* No. 31) confirms the sedative action of K. Br. on man, and the destruction of reflex sensibility of the pharynx. On an empty stomach the effects are more intense, but also the local action (producing diarrhœa, etc.). Dull headache, with loss of memory and impaired coordination of the movements of the tongue were usually observed as the effect of K. Br., but never sleepiness. Any action on the eye was not observed. In frogs, the voluntary muscles were finally paralyzed by K. Br., which paralysis was prevented by previous ligation of the blood-vessels. But even after cutting off the blood supply the motor nerves are in the end paralyzed, evidently a centrifugal paralysis. In rabbits, reflex sensibility was annihilated, before the occurrence of spasms (produced by cardiac paralysis). Hence K. concludes that reflex sensibility is destroyed before motor conductibility. Frogs can, therefore, perform voluntary movements when reflex movements are no longer possible. The dulling of cerebral sensibility K. compares to this annihilation of reflex power.

In order to estimate the share of either K. or Br. in these results, Klosz administered on the one hand a corresponding quantity of Na Br, on the other hand K Cl. The influence on pulse and temperature was equally possessed by K. Br. and K. Cl., much less by Na. Br.; it is hence referable mainly to potassium, which is also the active agent in the paralysis of muscle and nerve. The diminution of cerebral and reflex sensibility, on the other hand, is the result of the influence of bromine, due, no doubt, to a specific action of the nerve-substance. On the experience of Prof. Edlefsen, K. recommends small doses of K. Br. against unilateral neuralgia of the trigeminus.

Other recent papers on the Therapeutics of the Nervous System and Mind:

ROBERTS, Treatment of Traumatic Tetanus, *Am. Jour. Med. Sci.* Oct. 1877; LIEBREICH, Observations on the Use of Chloral, *Lancet*, (Am. repr.) October, 1877; BESNIER, Subcutaneous Injections of Chloroform, and particularly their Employment in the Treatment of Pain; *Bull. Gen. de Thérap.* No. 30; AYER, Result of the Brown-Séquard Treatment in Twelve Cases of Epilepsy, *Boston Med. and Surg. Jour.* Dec. 24; SEGUIN, A Contribution to the Therapeutics of Migraine, *New York Med. Record*, Dec. 8; ENGEL, The Diagnosis and Treatment of Some Forms of Syphilitic Nervous Affections, *Phil. Med. Times*, Dec. 22.